ACS Chemical Neuroscience

In This Issue

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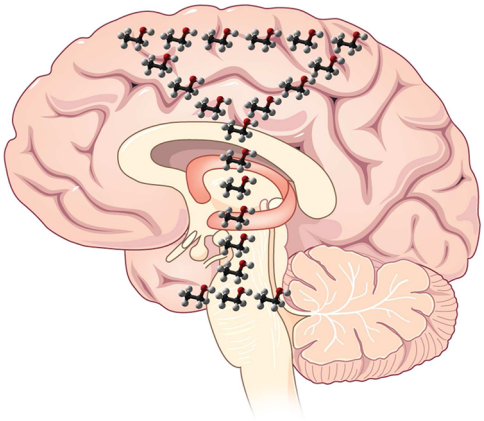
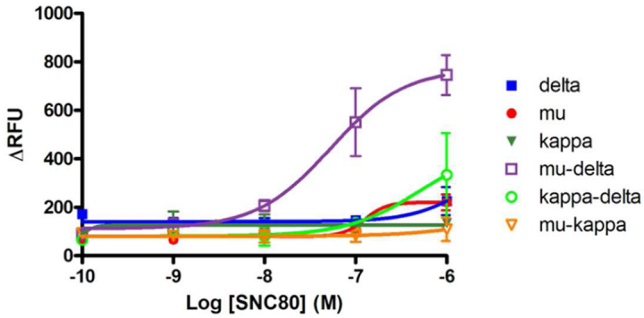
NEUROCHEMISTRY OF ALCOHOLISM

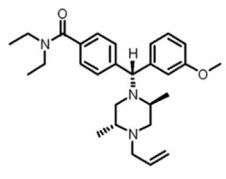


Alcoholism or alcohol addiction is a chronically relapsing disorder that is characterized by numerous changes in the chemistry and structure of the brain. A gap exists in our understanding of the changes in neurocircuitry caused by chronic alcohol consumption. Thus to date, few pharmacological compounds have been developed for alcoholism treatment. In this issue, Gass and Olive (DOI: 10.1021/cn300013p) provide an overview on the recent developments related to the neurochemistry of alcoholism.

The authors discuss chronic alcohol consumption induced changes to various chemical messenger systems in the brain and the decline in brain structures vital to neuronal communication. Recent studies in human and animal models within the context of alcoholism-induced cognitive and behavioral abnormalities have opened the door for new therapeutic opportunities.

SNC80 ACTIVATES $\mu-\delta$ HETEROMER



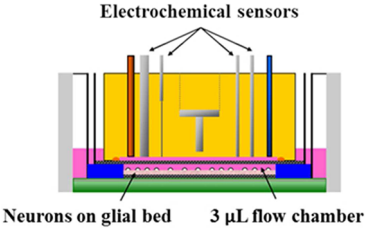


SNC80

Opioid receptors are a target for the development of analgesics. Over the last 20 years, SNC80 has been thought of as a selective δ-opioid receptor agonist. However, more recent studies have revealed that the majority of these G protein-coupled receptors are organized as dimers rather than as a single signaling unit. In the current issue, Metcalf et al. (DOI: 10.1021/cn3000394) revisit the selectivity of SNC80 and show that this compound in fact activates heterodimeric $\mu-\delta$ opioid receptors.

The authors utilized *in vitro* studies using a calcium fluorescence assay, opioid receptor knockout mice, and comparison of the antinociceptive effects *in vivo*. The startling observation that SNC80 exerts its effects through interaction with the $\mu-\delta$ heteromer has implications in the interpretation of data in experiments that employ this compound as a pharmacologic tool.

MONITORING NEURONAL METABOLISM IN REAL-TIME



There is a gap in our understanding of metabolic adaptation of primary neurons to stresses, such as nutrient deprivation. To bridge this gap, researchers have begun investigating various metabolites as predictors of neuron survival. In this issue, McKenzie et al. (DOI: 10.1021/cn300003r) use multianalyte microphysiometry to monitor the metabolic response to stress in real time.

The authors used multianalyte microphysiometry for simultaneous electrochemical measurements of glucose, lactate, oxygen, and acid. A link between metabolic recovery and neuronal survival was established. Additionally, this novel approach revealed that acid, rather than lactate, is a better predictor of neuronal survival. These results confirm the utility of studying multiparameter metabolites in real time, which may lead to future discoveries in post-stress neuronal survival.

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